Effect of Nasal Obstruction on Upper Airway Muscle Activation in Normal Subjects*

Stephen C. Wilhoit, M.E.; and Paul M. Suratt, M.D., F.C.C.P.

It is not known whether nasal occlusion produces obstructive sleep apnea (OSA) by decreasing upper airway muscle activation via nasal reflexes or by increasing upper airway resistance and hence lowering the pressure in the pharynx. The purpose of this study was to determine the effect of nasal occlusion on upper airway muscle activation. We studied seven men and measured alae nasi (AN) and genioglossal (GG) electromyograms (EMGs) during two nights of sleep, one with their nose open and the other with their nose occluded. Nasal occlusion produced OSA in all subjects and also increased the percentage of time during sleep in which phasic AN and GG EMG activity was present. Apneas tended to occur at the nadirs of EMG activity. This suggests that nasal occlusion generally increases respiratory drive to upper airway muscles during sleep and that it does not cause OSA by merely decreasing respiratory drive to these muscles.

Nasal obstruction has been shown by several investigators to produce sleep apnea in normal subjects.1-3 Although early studies suggested that the apnea was largely central,1 recent studies employing esophageal catheters have shown that it is predominantly obstructive.1-3

The mechanism by which nasal obstruction leads to obstructive sleep apnea (OSA) is unknown. It has been hypothesized that air flowing through the nostrils stimulates nasal reflexes, which in turn stimulate contraction of upper airway muscles to maintain a patent airway during inspiration.1 With nasal obstruction, airflow through the nose would stop and contraction of the upper airway muscles would decrease and produce an apnea. If this were true, one would expect that occluding the nose would decrease upper airway muscle activation during sleep.

Another possibility is that nasal obstruction produces obstructive sleep apnea by increasing upper airway resistance. For this to occur, mouth breathing during sleep would have to be a higher resistance pathway than nose breathing. Inspiring through the mouth would thus make the pressure in the pharynx more negative and tend to collapse the airway. Under these circumstances, upper airway patency would be maintained only if upper airway muscle activity was increased.

The purpose of this study was therefore to determine whether nasal occlusion during sleep in normal subjects decreased or increased activation of upper airway muscles.

**METHODS AND MATERIALS**

We studied seven men whose anthropometric characteristics are described in Table 1. Subjects were studied on two consecutive nights; on the first night, their nose was not occluded and on the second night it was. Since this investigation was performed as part of a study of prolonged nasal occlusion, nasal occlusion night was not randomized to the first or second night.

Nasal occlusion was produced by inserting strips of gauze impregnated with petrolatum into the anterior 2 cm of the nostrils. Subjects did not find the procedure painful and neither topical anesthesia nor sedation was necessary.

Electromyograms of the alae nasi (AN) and the genioglossus (GG) were measured using surface electrodes, as previously described.4 Electromyograms of the GG could not be measured in two subjects because they had beards. Electromyograms (EMG) of the genioglossus and alae nasi were recorded from silver chloride pellet surface electrodes (Graphic Control Corp, Rochester, NY). Genioglossus electrodes were positioned lateral to the midline, 1 cm apart and half-way between the chin and hyoid bone.4 Alae nasi electrodes were attached on the lateral inferior surface of the nose, as previously described.5 Signals from the EMG were amplified at band-pass frequencies of 10 to 300 Hz (Grass Inst). They were then full-wave averaged for 30-minute intervals.

**Table 1—Anthropometric Characteristics**

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<tr>
<th>Subject</th>
<th>Age (yrs.)</th>
<th>Ht (cm)</th>
<th>Wt (kg)</th>
<th>BMI* (kg/m²)</th>
<th>FVC† (%)</th>
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*Body mass index = patient’s weight in kg divided by the square of ht in meters; normal value is less than 27.
†Forced vital capacity.

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Figure 1. Nasal occlusion increased the number of apneas plus hypopneas per hour of sleep in all subjects. Numbers refer to subject number in tables.

Rectified and processed with an integrating filter on a 200 ms time constant to yield a moving time average.

Phasic respiratory activity of the EMG was considered to be present when there was increased activity of the raw EMG during inspiratory attempts. The entire sleep tracing was analyzed in all subjects and the number of minutes in which phasic activity was present was expressed as a percentage of total sleep time and also REM sleep time in minutes.

In each subject, we also measured alae nasi EMG signals during six apneic episodes in detail during the nasal occlusion night. Amplitude of the moving time average was measured for three breaths prior to the apnea, for the first and last three inspiratory attempts during the apnea and for the first three breaths following apnea.

Nocturnal sleep studies were performed using standard techniques including measurement of EEG (C3-A1, C3-O1), EOG, submental EMG, electrocardiogram, oxygen saturation, esophageal pressure and airflow with thermistors placed within a loosely-fitted facemask. Sleep was manually staged according to standard criteria by experienced technicians. The degree of sleep apnea was quantitated by analyzing the airflow tracing.

Data were analyzed with the Wilcoxon test.

Results

Nasal occlusion produced obstructive sleep apnea in all patients (Fig 1). In all subjects, the percentage of time in which phasic activity in both alae nasi and genioglossus EMG was present was greater when the nose was occluded than when it was not occluded (p<0.05) (Figs 2, 3).

Phasic alae nasi activity was present at apnea onset in six of seven subjects, although the magnitude of activity decreased at apnea onset in five of the seven subjects (Fig 4).

Discussion

This study has shown that nasal occlusion which produces obstructive sleep apnea in normal subjects increases phasic EMG activity of the AN and GG throughout sleep. Phasic activity was present even at apnea onset in most subjects, although the magnitude of change was decreased from immediately prior to the...
Figure 4: Alae nasi EMG activity during the three breaths before apnea (1-3), the first three (4-6) and last three (7-9) inspiratory attempts during apnea, and the three breaths immediately following apnea (10-12). Subject number is in upper right hand corner of each graph. Phasic activity was present at apnea onset in all subjects except number 5. The magnitude of EMG activity decreased at apnea onset in all subjects except 5 and 6.

Apnea. Apnea did not occur during the unoccluded night, even when there was no phasic activity.

The increase in phasic EMG activity during sleep suggests that nasal occlusion augments respiratory drive to upper airway muscles. Apneas tend to occur, as in patients with OSA, when there is a decrease in the magnitude of this drive.

These findings suggest that nasal occlusion does not cause OSA by decreasing respiratory drive to the upper airway. However, the findings are consistent with the hypothesis that nasal occlusion forces subjects to breathe through a high resistance pathway—the mouth—during sleep. Breathing through a high resistance pathway would make the pressure in the pharynx more negative and thus requires more upper airway muscle activation to maintain airway patency. Apneas would occur when there is insufficient muscle activation to offset the more negative pharyngeal airway pressures.

It is also possible that nasal occlusion directly stimulates upper airway muscles through an unknown reflex. However, this would not explain why nasal occlusion produces OSA. It would suggest, rather, that nasal occlusion should protect against OSA.

Nasal occlusion has not been shown to decrease hypoxic and hypercapnic ventilatory drive and may even increase it. This suggests that OSA can be produced by solely increasing airway resistance without decreasing CNS drive. If this is true, nasal occlusion could serve as a model of OSA produced by a narrow airway alone.

We measured genioglossus and alae nasi EMG activity with surface electrodes since they are more practical and less painful than needle electrodes. Fine wire or needle electrodes inserted in the body of the genioglossus would undoubtedly have improved our sensitivity and specificity. Since, however, we used the same techniques in all subjects on both nights, the differences we observed between unoccluded and occluded nights are unlikely to be due to artifact or recording technique. Also, differences in EMG signals due to recording technique would be expected to produce random results, whereas we did observe significant differences between the two experimental conditions.

References
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